An Opponent-Process Theory of Motivation[1, 2, 3]

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*The paper by Solomon and Corbit is regarded by many psychologists as the most successful attempt so far of providing a general theory capable of explaining both psychological addiction and some people's acquired taste for exposing themselves to extreme danger. Apart from the omission of two somewhat technical sections, in which the authors relate their hypothesis to Pavlovian conditioning, the paper is reprinted in full. It is the first of a series of articles: the one which presents the hypothesis and gives an overview of its applications. The subsequent articles contain accounts of experiments designed to test the hypothesis.**

*The behavioral hypothesis is clearly one the postulate of consumer rationality cannot very well explain. Addiction, like externalities and non-convexities, is one of those awkward exceptions that do not fit into the economist's standard models. We might, perhaps, be excused for ignoring it, if addiction were confined to a few chemical substances and due to physiological causes; but there is a growing suspicion of its being a much more general phenomenon. The psychologists' motivation theory has a large overlap with our theory of consumer preference; the Solomon and Corbit article has been chosen as a good example of their approach to a problem we have not yet considered though are likely, sooner or later, to feel compelled to consider. The approach of the article differs from and should supplement that of the more general theory of motivation which is built around the concept of arousal and formed the core of Scitovsky's account of the psychologists' view of individual behavior.***

T. Scitovsky

First, we describe the kind of phenomenon which has caught our attention. Two fictitious examples will suffice. In the first, a woman at work discovers a lump in her breast and immediately is terrified. She sits still, intermittently weeping, or she paces the floor. After a few hours, she slowly regains her composure, stops crying and begins to work. At this point, she is still tense and disturbed, but no longer terrified and distracted. She manifests the symptoms usually associated with intense anxiety. While in this state she calls her doctor for an appointment. A few hours later she is in his office, still tense, still frightened: She is obviously a very unhappy woman. The doctor makes his examination. He then informs her that there is no possibility of cancer, that there is nothing to worry about, and that her problem is just a clogged sebaceous gland requiring no medical attention.

A few minutes later, the woman leaves the doctor's office, smiling, greeting strangers, and walking with an unusually buoyant stride. Her euphoric mood permeates all her activities as she resumes her normal duties. She exudes joy, which is not in character for her. A few hours later, however, she is working in her normal, perfunctory way. Her emotional expression is back to normal. She once more has the personality immediately recognizable by all of her friends. Gone is the euphoria, and there is no hint of the earlier terrifying experience of that day.

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*R. L. Solomon and J. D. Corbit, *Journal of Abnormal Psychology*, Vol. 81, pp. 158–71, 1973.

Howard S. Hoffman and R. L. Solomon, *Learning and Motivation*, Vol. 5, pp. 149–64, 1974.

R. L. Solomon in *Psychopathology: Experimental Methods*, edited by J. D. Maser and M. E. P. Seligman, 1977, pp. 66–103.

R. L. Solomon in *Learning Mechanisms in Food Selection*, edited by L. M. Barker, M. R. Best, and M. Domjan, 1977.

M. D. Starr, *Journal of Experimental Psychology*, 1978 (in press).

**Cf. T. Scitovsky, *The Joyless Economy* (O.U.P., 1976, New York) chapters 2 through 4.

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[2]A subtitle, "I. Temporal Dynamics of Affect," and an abstract were omitted in this reprinting.

[3]Reprinted by permission from *Pschological Review*, Vol. 81, No. 2, pp. 119–133, 142–145, 1974.

In the second example, a couple have just begun sexual foreplay, and it is quite pleasurable. After a few moments of a constant level of mutual stimulation, the pleasure decreases somewhat. Normally, this decline would elicit behavior calculated to increase the intensity of mutual stimulation and to maintain the high level of pleasure. Unfortunately, at that moment a telephone rings. One partner leaves and goes into another room to answer it, and the other partner lies alone the in bed. The abandoned partner experiences a quick decline of the pleasure, then becomes tense and irritated, and strongly craves a resumption of the sexual stimulation. Time goes by, however, and the other partner does not return. Finally, the abandoned partner gets out of bed, absentmindedly turns on the television set, and becomes absorbed in a news broadcast. Gone is the irritability and intense craving. There is no hint, in overt behavior, of the pleasurable sexual experience of a few minutes ago. A type of dispassionate normality now pervades.

We can distill from these two examples some important empirical features common to many hedonic, emotional, or affective experiences. First, following the sudden introduction of either a pleasurable or aversive stimulus, an affective or hedonic reaction begins and quickly rises to a peak. It then slowly declines to a steady level where it remains if the stimulus quality and intensity is maintained. Then, at the sudden termination of the stimulus, the affective reaction quickly disappears and gives way to a qualitatively very different type of affective reaction which reaches its own peak of intensity and then slowly disappears with time.

Figure 1 diagrams these changes in hedonic or affective state and illustrates what we call the *standard pattern of affective dynamics*. The pattern has five distinctive features: (*a*) the peak of the primary hedonic process or state, precipitated by stimulus onset; (*b*) a period of hedonic or affective *adaptation* during which the intensity of the hedonic state declines, even though stimulus intensity is maintained; (*c*) a *steady level* of the hedonic process which continues as long as stimulus intensity is maintained; (*d*) a *peak of affective after-reaction,* which quickly follows stimulus termination, and whose quality is hedonically very different from that of the primary hedonic state; and (*e*) finally, the afterstate decays and subsequently disappears.

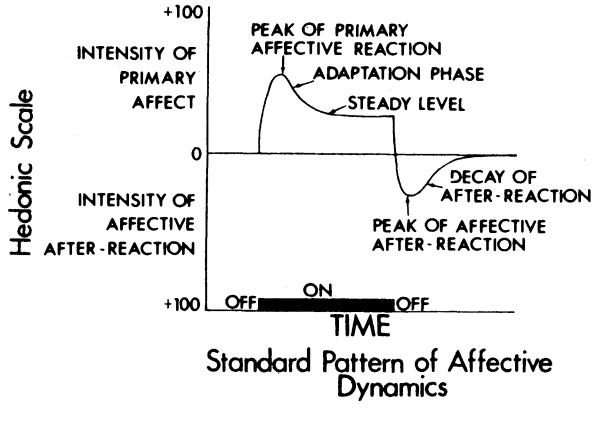

FIGURE 1. The standard pattern of affective dynamics, showing the five distinctive features: the peak of the primary affective reaction, the adaptation phase, the steady level, the peak of the affective after-reaction, and, finally, the decay of the after-reaction. (The heavy black bar represents the time during which the affect-arousing stimulus is present. The ordinate represents two hedonic scales, each departing from neutrality, one for the primary affect, the other for the affective after-reaction.)

This standard pattern describes both fictitious examples. In the first, the initial, primary hedonic or affective process was unpleasant and the after-reaction was pleasant. In the second, the primary process was pleasant and the after-reaction was unpleasant. The data of psychology contain literally dozens of examples of this kind. For brevity we will first describe some of the more interesting or important cases chosen from very different areas of psychological research. We can therefore demonstrate the great generality of the standard pattern of affective dynamics. Then we will describe a theoretical model for the underlying mechanism.

Examples of Empirical Phenomena To Be Explained

Table 1 presents seven examples of emotional, affective, hedonic, or motivational phenomena. Some are "behavioral," some "experiential." Some are experimental, others are observational, derived from common everyday experiences. Finally, some are precipitated by pleasant, and some by unpleasant stimuli.

Example 1 describes behavior changes seen in a dog subjected to intense aversive stimulation. The example draws on selected parts of studies reported by Katcher, Solomon, Turner, LoLordo, Overmier, and Rescorla (1969) and Church, LoLordo, Overmier, Solomon, and Turner (1966). A dog in a Pavlov harness was stimulated by several 10-second shocks. The dog appeared to be terrified during the first few shocks. It screeched and thrashed about, its pupils dilated, its eyes bulged, its hair stood on end, its ears lay back, its tail curled between its legs. Expulsive defecation and urination, along with many other symptoms of intense autonomic nervous system activity, were seen. At this point, the dog was freed from the harness, it moved slowly about the room, appeared to be stealthy, hesitant, and unfriendly. Its "state" had suddenly changed from terror to stealthiness.

We now arbitrarily label the state during shock (the terror state or the peak of the primary reaction to shock) State "A." The stealthy state, right after shocks were terminated (the after-reaction), will be called State "B." In that way, we can temporarily ignore whether we have correctly labeled the states. We know that State A was not State B. Indeed, State A was very

TABLE 1

SELECTED EXAMPLES OF HEDONIC-AFFECTIVE PHENOMENA

	First few stimulations		After many stimulations	
Example	State A (input present)	State B (input gone)	State A′ (input present)	State B′ (input gone)
Dogs in Pavlov harness, 10-second shocks, gross behavior	terror, panic	stealth (subdued, cautious, inactive, hesitant)	unhappy (annoyed, anxious, afraid)	joy (euphoric, active, social), happy
Dogs in Pavlov harness, 10-second shocks, electrocardiograph responses	large cardiac acceleration	slow deceleration, small overshoot	small acceleration or none	quick decleration, large overshoot
Epstein's parachutists, free fall, gross behavior, physiology	terror, autonomic nervous system arousal	stunned, stony-faced	tense, eager, expectant	exhilaration, jubilation
Opiate users, intravenous injection, moods and feelings	euphoria, rush, pleasure	craving, aversive withdrawal signs, short duration	loss of euphoria, normal feeling, relief	intense craving, abstinence agony, long duration
Dogs and M & Ms, gross behavior	pleasure, tail wagging, chewing	tenseness, motionless	—	—
Love, interpersonal stimulation, moods, feelings	ecstasy, excitement, happiness	loneliness	normal, comfortable, content	grief, separation syndrome, long duration
Imprinting, the attachment of creatures to their "mothers"	pleasure, cessation of fear, no distress	loneliness, distress cries, short duration	pleasure, no cries	loneliness, intense cries, long duration

different from State B, judging by the many behavioral changes observed when the dog was suddenly released. Furthermore, State B gradually disappeared. In a few minutes, the dog appeared to be normal, like its previous, preshock, natural, self: active, alert, and socially responsive. When this happened, it was impossible to tell by looking at the dog that either State A or B had just transpired. The evidence was gone. The dog had progressed from "normalcy" to A to B, and back to "normalcy." This sequential pattern will be seen in all of the examples given in Table 1. *It is, we believe, the basic pattern for the dynamics of affect.*

However, Example 1 has not yet been fully described. When the same dog was brought back for the same treatment day after day, its behavior gradually changed. During shocks, the signs of terror disappeared. Instead, the dog appeared pained, annoyed, or anxious, but not terrified. For example, it whined rather than shrieked, and showed no further urination, defecation, or struggling. Then, when released suddenly at the end of the session, the dog rushed about, jumped up on people, wagged its tail, in what we called at the time "a fit of joy." Finally, several minutes later, the dog was its normal self: friendly, but not racing about. Here again, the sequence was normal→ State A→State B→normal, where State A accompanied the arousing input, and State B directly followed the sudden termination of that input, and then slowly died out.

We wish to emphasize that the qualitative and quantitative features of States A and B during *later* shocks were *not the same* as those for States A and B during the first few shocks. This pattern of changes, occurring as a consequence of repeated exposures to the stimulus input which causes A, also is, we believe, typical of the basic pattern of affective dynamics. Because the later States A and B were *not* identical to the earlier states, we label the later ones A′ and B′, respectively.

Look at Example 2 in Table 1. A dog was in an experiment in which its heartbeat was measured by an electrocardiograph during repeated shocks to its hind feet. The observations are from Church et al. (1966). During the first few shocks, there was a large cardiac acceleration; in some dogs it was an increase of 150 beats per minute. At shock termination, the rate suddenly decreased, and within 5 seconds it descended below the baseline rate. It often fell as low as 20 to 30

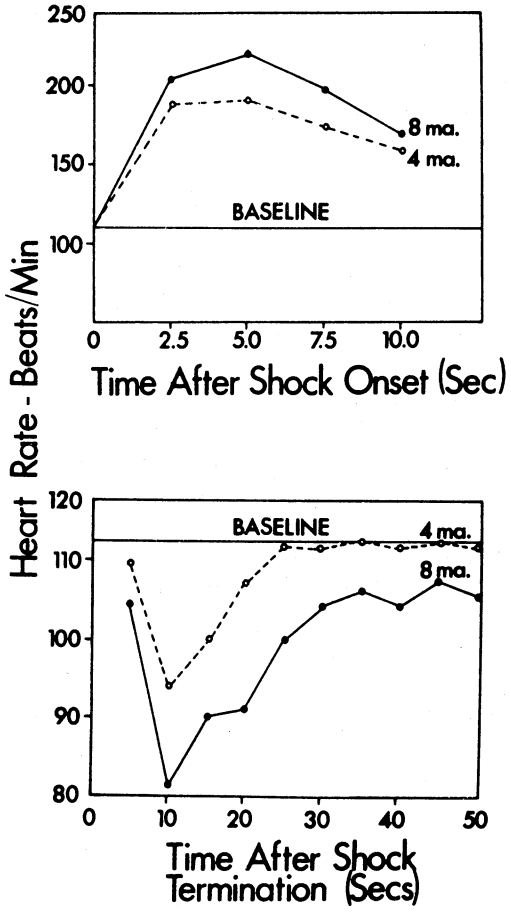

FIGURE 2. Heart rate changes as a function of shock onset, maintenance, and termination. (There is a decline following the initial peak reaction to shock onset. There is a decelera-tory "overshoot" following shock termination, and then the heart rate slowly returns to baseline rate. Note that the eight-milliampere shock produces a bigger heart rate increase and a bigger deceleration than does the four-milliampere shock.) (Adapted from an article by Russell M. Church, Vincent LoLordo, J. Bruce Overmier, Richard L. Solomon, and Lucille H. Turner appearing in the August 1966 *Journal of Comparative and Physiological Psychology*. Copyrighted by the American Psychological Association, Inc., 1966.)

beats per minute below baseline rate. Then, it slowly "recovered" to baseline rate, over a period of as long as 30 to 60 seconds. The below-baseline excursion has been called "vagal overshoot." It is a well-studied phenomenon. Figure 2 is taken from Church et al. (1966) and demonstrates these dynamic events. The figure also suggests that there may be a relationship between intensity of the A state and the magnitude and duration of the B state. Now we can define baseline, State A, State B, and the return to baseline using an electrocardiograph measurement instead of gross observations of emotional behavior.

States A and B changed markedly after several sessions. Shock onset now caused little, if any, increase in heart rate. Any increase was momentary. The rate often decreased even while the shock was still on. However, when shock was suddenly terminated, the "overshoot" was much larger than it was on early shock trials. Heart rate in some individual cases descended to 50 to 60 beats per minute (sometimes more than 50 beats per minute below baseline rate), and recovery to baseline took as long as two to five minutes. As Katcher et al. (1969) put it, "The deceleratory heart-rate overshoot produced by stimulus termination shows shortened latencies . . . and greater magnitude over trials [p. 172]." Thus, States A and B have changed: A′ seems to be weaker than A, B′ stronger than B, and B′ longer lasting than B. But, as in the previous examples, we can still identify the sequence: baseline→A→B→baseline.

Example 3 comes from Epstein's (1967) report of physiological, emotional-expressive, and social reactions of parachutists. When parachutists make their first jump, they are often terrified, judging by telemetered autonomic responses and photographed facial expressions. When they land safely, they look stony-faced or stunned for several minutes, then gradually resume normal composure. After the parachutists have made several jumps and are experts, their responses are different. When jumping, they are no longer terrified. They may be anxious, tense, or even eager. After they land safely, they feel exuberant, exhilarated, and good. They like the feeling, and the mood lasts sometimes for hours. Such parachutists love to jump because of this after-feeling. Again, we see that the qualitative and quantitative attributes of States A and B have changed with the repetition of eliciting conditions. However, two very different states are still observable, and with each stimulation input, we can identify the sequence: baseline→A→B→baseline.

Example 4 represents states in opiate use (Jaffe, 1965; Maurer & Vogel, 1967). Early in a history of opiate use, the user experiences the "rush" (an intensely pleasurable feeling) directly after the opiate injection, followed by a period of less intense euphoria. Then, with further passage of time, the user suffers aversive, painful, and frightening somatic withdrawal symptoms, together with a feeling of craving. Here again, we see a baseline→A→B→baseline sequence. However, with opiates, B may last a long time, sometimes for several days.

After repeated dosages of opiates over several weeks, State A begins to weaken, and at the same time State B begins to intensify and takes longer to return to baseline. State A′ is called "normal" rather than euphoric. The rush is no longer experienced. Yet, State B′ is more physiologically disturbing than B was, and lasts much longer than did State B. The craving aspect of State B′ is now extremely intense, aversive, and enduring. It is called *abstinence agony*. It can be months before B′ returns to baseline. Perhaps it never really does. It is a ghastly experience.

The repeated use of some drugs results in the behavioral phenomenon of addiction. People find

themselves craving a substance in which they previously had little interest. It is the most vivid instance of acquired motivation, because of its intensity and duration. It also provides a fruitful, empirical model for analyzing many kinds of acquired motivation. Indeed, we later show, with examples taken from love and affection, social attachment, and imprinting in birds, that addiction does not differ in principle from *any* acquired motivational system. We can easily describe opiate, alcohol, barbiturate, amphetamine, or cigarette addiction (see Solomon & Corbit, 1973) within the empirical framework of the analysis we have proposed. They all have four attributes: (*a*) The B′ state lasts a long time; (*b*) the B′ state is intensely aversive; (*c*) the elicitation of State A or A′ is effective in causing immediate removal of State B or B′; and (*d*) the user learns to employ the drug which elicits States A and A′ in order to get rid of State B or B′.

A lasting cycle of addiction will *not* arise, even though A and B are repeatedly experienced, if the properties of affective response to a drug are such that B fades out to baseline very quickly. This is true because another dosage of the drug is never needed in order to get rid of the aversive B state. It quickly gets rid of itself. This is the case in Example 5, a common type of pleasurable situation. A laboratory dog is sitting dejectedly in a cage. It is suddenly handed one M & M candy. It wags its tail, moves about, chews, and swallows. This reflects State A. It smacks its lips a few times, curls its tongue across its lips, then becomes motionless and tense. It orients toward the experimenter and focuses its eyes on the experimenter's hand (the one which held the single M & M). Assume this to reflect State B. Then, if another M & M is not forthcoming, the B state dies out after about 15 to 30 seconds. The dog moves away, walks around the cage, sniffs here and there, begins to "ignore" the experimenter, then sits again. It has returned to its original state. Here again, termination of one hedonic event has precipitated another state not present prior to the onset of stimulation, and the second state disappeared by itself merely with the passage of time. In this case, A is pleasant, so we infer B to be qualitatively different, and probably *craving* is the best term for it. The so-called "peanut phenomenon" in humans is comparable. Once you start eating peanuts, it is hard to stop unless the cycle is interrupted for a period longer than the time required for the peanut craving, or B state, to die out. This has been called a mini-addiction.[3] Indeed, the case of the couple interrupted during mutual sexual stimulation, described in the Introduction, is certainly similar.

In Example 6, the pleasurable input generates a condition in which the B state typically lasts a lot longer than that for an M &M or a peanut, and so the favorable conditions for addiction are present. A boy and girl "fall in love." This State A is characterized by pleasurable excitement, frequent sexual feelings, a prevailing mood of ecstasy, happiness, and good feelings. When the lovers, whose multimodal mutual stimulation will cause State A, are separated from each other, they will feel lonely, sad, and depressed (State B). Even with anticipations of reunion (symbolic, conditioned arousers of State A) loneliness may prevail. Actual reunion will simultaneously erase B and reinstate A just as described in Attribute *c* of addiction to opiates.

After several years of repeated mutual stimulation, the qualitative and quantitative changes in A and B are a matter of public lore. State A′ is characterized (if all has gone well) as contentment, normalcy, and comfort. But State B′ is now potentially of high intensity and long duration. If it should occur, it is often called grief or, as Bowlby (1952) has described it in children, the "separation syndrome." It requires a lot of time for this B′ state to decay. The partners have become addicted to one another, and when separated they experience withdrawal symptoms. As in all the previous five examples, note that the sudden termination of the stimulus that arouses A or A′ leads to the occurrence of B or B′ before the eventual return to emotional baseline or normalcy. In this case, the termination of A′, even though A′ does not manifest itself as intensely as did A, is followed by a more powerful and much more protracted B′ state. This is the same pattern seen in all the previous examples, whether the A state is pleasurable or aversive.

Example 7 is one of imprinting. If we take the duckling as our subject, it shows the same patterns of affect revealed in opiate addiction. First, right after hatching there are very few distress cries. Indeed, the duckling may appear to be quite happy with its new environment. Then, if the duckling is exposed to a white, moving object, it looks intently at it. Also, if there were any distress vocalizations, they tend to disappear. However, if the moving object is then removed from view (Hoffman, 1968; Hoffman, Stratton, Newby, & Barrett, 1970), there is a burst of distress crying which may last for several minutes and then disappear. With successive presentations and removals of the imprinting stimulus, the frequency and intensity of distress crying will increase.

The efficacy of the presentation of the moving stimulus in eliminating the distress cries appears to be optimal at the outset. Then the duckling can be shaped to push a pole in order to present itself with the imprinting stimulus. At that point, of course, the duckling is exhibiting all criteria for addictive behavior. It is "hooked" on the imprinting object, the presence of which is a positive reinforcer and the absence of which is an aversive event.

Note that the ethological description of imprinting as the sudden establishment of "following behavior,"

[3]This term was first suggested by Eliot Stellar at a cocktail party.

released by an adequate imprinting stimulus, is utterly inappropriate in the light of our analysis. The moving object releases some affective State A, presumably an unconditioned, pleasant emotional reaction to the moving stimulus. The removal of the stimulus then precipitates State B, which is an aversive event. State B intensifies with repeated stimulations. Ducklings will then work on an avoidance schedule to prevent the disappearance of the imprinting object, just as the opiate addict will develop anticipatory behavior which prevents the occurrence of at least the more intense withdrawal symptoms.

Empirical Generalizations

First, in all seven empirical examples, as well as in the fictitious ones in our Introduction, the sudden onset of some new stimulus aroused an affect or hedonic state not present prior to onset. The state terminated when the stimulus terminated. Then, a new state appeared, *qualitatively unlike* either the prestimulation state or the state produced by the onset and maintenance of the stimulus. Finally, this new poststimulus state persisted for a while and died out. The baseline state eventually returned. In none of the examples did the subject's affective state return directly to baseline upon cessation of stimulation. Baseline was regained via some new state which became manifest at stimulus termination, and then slowly died away.

Second, in some cases the states changed in their quality and intensity with successive, repeated stimulations. Whenever this occurred, the A states became weaker and the B states stronger and longer lasting.

These two phenomena, the dynamic hedonic response pattern, and its modification with repeated experience, were seen whether the A state was pleasurable or aversive.

The Explanatory Model

In our opinion, the simplest theoretical model that organizes these typical motivational phenomena (and countless other phenomena of innate and acquired motivation) is an opponent-process system. It was borrowed from previously developed accounts of sensory dynamics (Hurvich & Jameson, 1957). The primary *a* process for a given hedonic state is aroused by its adequate stimulus. We then imagine a single opponent loop generating the secondary *b* process and having an hedonic sign *opposite* to that of the state aroused by the input. The loop generating the *b* process is activated whenever any input evokes a sufficient hedonic consequence. The *b* process is sluggish, so it has a relatively long latency, recruits slowly, and dies out slowly. Finally, the *b* process is strengthened by use and weakened by disuse.

Because many formal properties of the patterns of both sensory and affective phenomena seem so similar, we must show at the outset that they are *not* identical and that the standard pattern of affective dynamics is *not* a direct consequence of the pattern of sensory dynamics. Otherwise, one could explain all hedonic or affective dynamics in terms of sensory events. For example, in the case of the woman who discovered the lump in her breast, one might argue that her steady-level anxiety state was less intense than her initial peak of terror at the discovery of the lump because of sensory adaptation; that is, the perceived presence of and magnitude of the lump decreased. We can refute such an interpretation, leaning heavily on the fact that the *time course* of typical sensory dynamics is of a completely different order of magnitude from that of the standard pattern of affective dynamics reflected in Figure 1 and Table 1. Sensory changes usually occur in a matter of milliseconds, seconds, and minutes, whereas the emotional changes usually occur in minutes, hours, days, weeks, and months. There is, therefore, a theoretical necessity for a distinct opponent-process mechanism for affect and motivation independent of mechanisms for sensory dynamics.

Figure 3 illustrates the type of system that we have in mind. Panel A shows two stages of information processing, a cognitive-perceptual stage that converts the stimulus to an informational signal, and an affective or hedonic stage that converts the informational signal to an affective signal. The affective system in Figure 3 receives a square wave input, and follows it with a dynamic affective response of the standard pattern shown in Figure 1.

Perhaps an example will clarify the point of Figure 3. The sight of a dog is a fear-arousing stimulus for a cat. The dog represents a complex, multidimensional display, and the perception of the dog is categorical, all-or-none. The stimulus sequence is as follows: The dog enters the cat's environment, remains for a while, and then leaves. The cognitive-perceptual sequence is: The dog is detected, continues to be detected as long as it remains, and then ceases to be detected when it leaves. There is no adaptation (i.e., the dog does not become less doglike), nor is there any appreciable sensory after-reaction at stimulus removal (i.e., no negative [antidog?] afterimage). The affective sequence, in contrast, will show the primary reaction and after-reaction components: intense fear at first, subsiding to a steady level, and then, when the dog is gone, the appearance of another state, very different from the first. The after-reaction (relief?) then gradually dies out.

Figure 3, Panel A, shows that the affective system follows its square wave input with an output having the complex dynamic features of Figure 1. How can we account for this behavior? The affective system in Panel A is shown as a single stage. Now, we will open the "black box" for this stage and look inside. Our proposal for the mechanism responsible for affective dy-

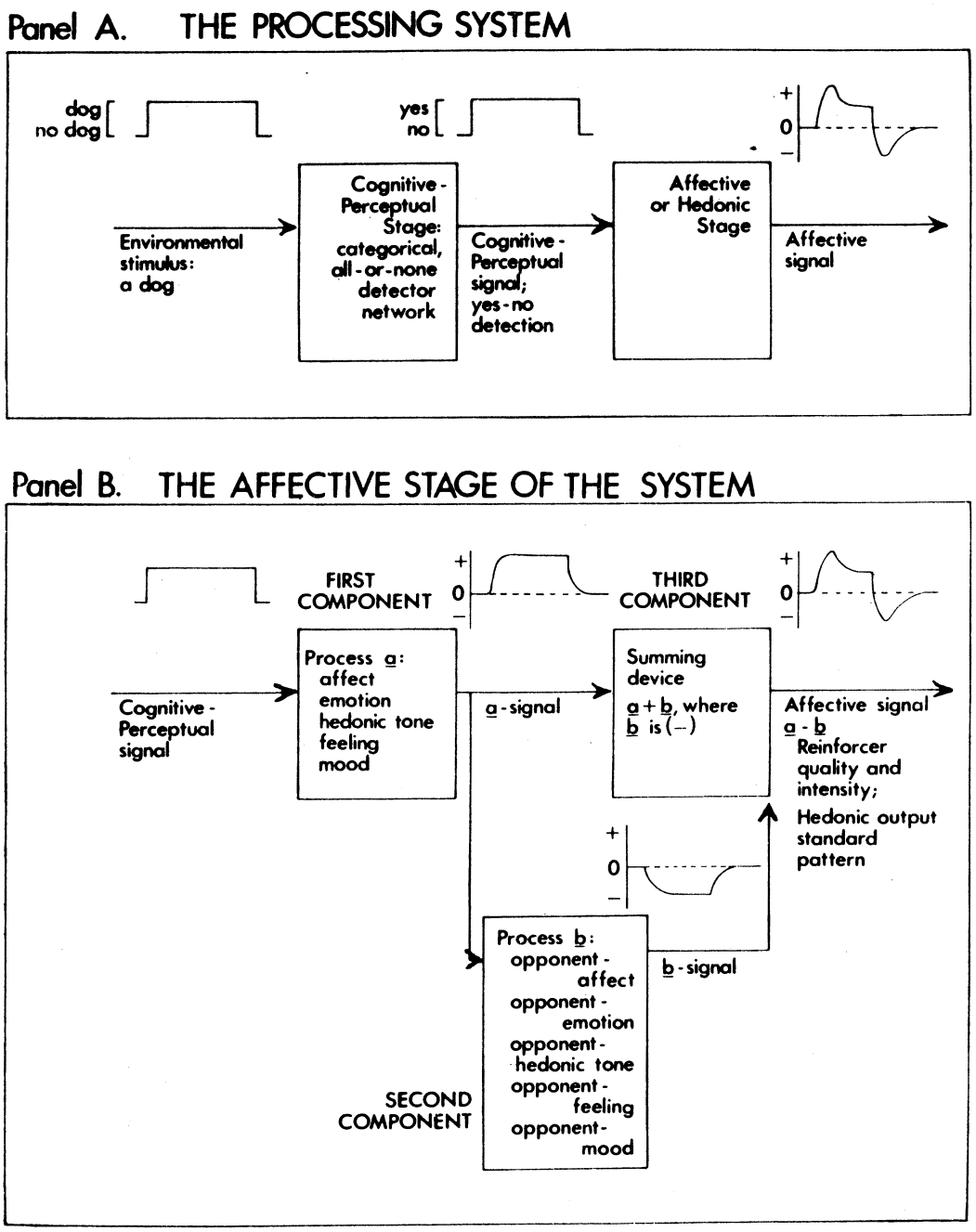

FIGURE 3. Panel A: The detection of an environmental stimulus by a cognitive-perceptual mechanism, and the arousal of an affective stage yielding an affective signal which shows the standard pattern of Figure 1. Panel B: An analysis of the three components of the affective mechanism. (In the first component the *a* process is aroused. The second component, the *b* process, is aroused via the arousal of *a*. Then the third component, a summing device, combines the *a* and *b* signals to generate the standard pattern of affective dynamics.)

namics is shown in Figure 3, Panel B. Here, the affective stage is analyzed into its three component parts. The cognitive-perceptual stage has acted as a categorical detector. The informational signal enters the affective system as input to the first component, the *a* process, which has a short time constant. The signal from the *a* process activates the second component or *b* process, which responds with a slow rise and a slow decay. The third component is a summing device that adds the *a* and *b* signals, and it generates as its output the affective signal, which shows the sequence of the peak primary reaction A, adaptation, steady level, after-reaction B, and decay of B. Thus, we see that the input from the perceptual-cognitive stage has an affective, hedonic side effect, Process *a*. When it does, the opponent loop is activated, calling into play the opponent process (which has a hedonic quality in some way opposite to, and very different from, that of Process *a*). The opponent process, which we call Process *b*, reduces the hedonic intensity of the state which the input ini-

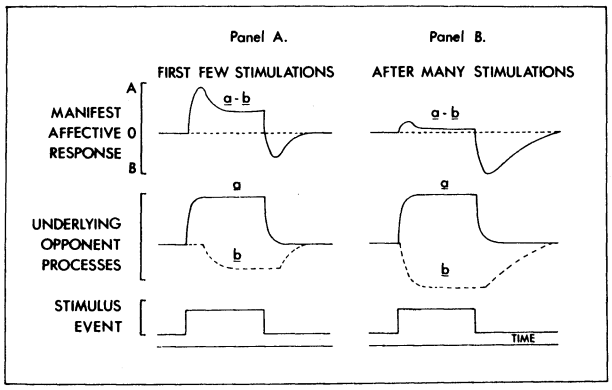

FIGURE 4. Panel A: The operation of the summing device for the first few stimulations. (The summation of the underlying opponent processes, *a* and *b*, yields the manifest affective response.) Panel B: The operation of the summing device after many repeated stimulations.

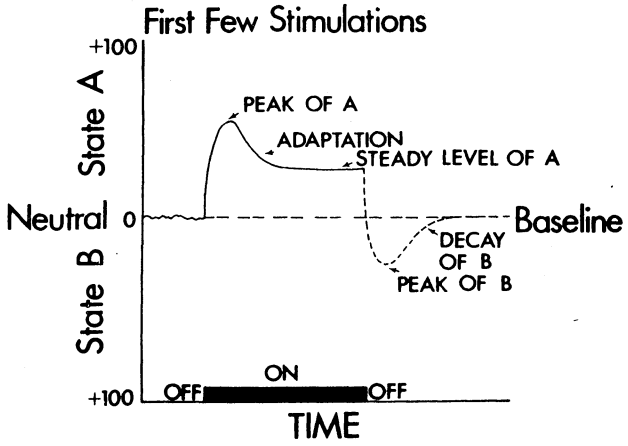

FIGURE 5. The manifest temporal dynamics generated by the opponent-process system during the first few stimulations. (The five features of the affective response are labeled.)

tially aroused. When the perceptual-cognitive input ceases, the opponent process reveals itself as "pure" State B, because the *b* process takes a while to decay.

The opponent process is a *slave process:* It is activated indirectly via the activation of the *a* process. Presumably, the slave process has an evocation threshold, a latency, a recruitment or augmentation time, and a decay function, all characteristic of a given opponent-process system. We will also see later that the opponent process can, under proper conditions, be activated by events in memory, as a consequence of Pavlovian conditioning procedures.

The block diagram of the affect-control system in Figure 3 yields the temporal dynamics of affect shown in Figure 4, Panel A. There we first see a baseline state. Then the affect-arousing stimulus is presented and it stays on for 10 seconds. Next, it is suddenly terminated. This simple event sequence activates the underlying opponent processes. First, there is a quick rise of Process *a* to a peak intensity. Shortly afterward, there is a slow recruitment of Process *b*. When the stimulus is terminated, Process *a* quickly goes to zero, but Process *b*, having a sluggish decay property, perseverates and dies out slowly. The resultant manifest dynamics of affect are a consequence of subtracting the *b* process from the *a* process. The subtraction of the two quantities yields Manifest State A when $a > b$ and yields Manifest State B when $b > a$. The intensity of the manifest state is given by the quantity $|a - b|$.

When we subtract the underlying processes, *a* and *b* from each other, we obtain the manifest affective response pattern shown in Figure 5. Immediately after stimulus onset, *a* is large and *b* is zero, thus yielding a peak of State A. As *b* is slowly recruited, the quantity $|a - b|$ decreases, yielding a decline in the magnitude of State A. This is the adaptation phase shown in Figure 1, the standard pattern of affective dynamics. When the *b* process reaches an asymptote, there will be a steady level of State A. Then, when the stimulus is terminated, the intensity of *a* goes quickly to zero, but the *b* process dies away slowly. At this moment, $b > a$, yielding the peak and subsequent decay of State B. At this point, State B is pure *b*.

The theory represented in Figures 3 and 4 gives a rough, qualitative account of all of the data of Table 1, for the first few stimulations only. First, it explains peaks of intensity of affect or hedonic quality at stimulus onset: The opponent process has not yet had enough time to get into action. So we see phenomena like terror, the rush, ecstasy, etc. Or, rate the painfulness of a 30-second shock. The peak painfulness is at onset. For example, see the peak of heart rate in Figure 2.

The other major event explained by the theory is the emergence of the *after-reaction,* postulated to be a function of the opponent process, which becomes manifest after the termination of stimulus input. This emergence is due to pure *b* perseverating in time after the *a* has quickly disappeared. The B state slowly decays, and baseline is eventually attained.

In each of the seven empirical examples of Table 1, the manifest B state was, in some unspecified way, related to A, but not the same as A. The model designates the relationship as *oppositeness.* But in what way can we say, for example, that loneliness is opposite to the pleasure produced by the presence of a loved one? Surely, they are hedonically opposed. This concept is built into the theoretical model. If A is pleasant, then B must be unpleasant. If A is a positive reinforcer, then B is a negative reinforcer. Other affective attributes of A and B remain an empirical question. The model tells us how to identify the attributes of oppositeness. All one has to do, for any given A state, is observe the attributes of affect which are revealed at the peak of B. Therefore, the model puts a constraint on what we call oppositeness. For example, the opposite of love must be the attributes of grief. To say that "Hate is the oppo-

site of love" is simply wrong, when at the death of a loved one, one experiences grief, not hate. The kinds of oppositeness which are generated by each A state are still, in many cases, waiting to be studied and named. Here is a vast, neglected area of investigation.

We postulated that the *b* process is a slave process. That means that at first it cannot be aroused directly by ordinary sensory inputs, but instead can arise only *indirectly* via the arousal of an *a* process (see Figure 3) and the subsequent activation of the opponent loop. This fits common sense. Try to imagine being grief-stricken without having loved someone. Try to imagine craving and abstinence agony without drug use. Try to imagine the exhilaration of the parachutist without any jump. On the other hand, we will point out later that it may be possible to arouse a *b* process directly by electrical or chemical stimulation of the brain, or to eliminate it by surgery, or to condition it by Pavlovian procedures. Initially, however, it is a slave process, inaccessible to direct environmental inputs, but indirectly arousable via hedonic and affective processes elicited by environmental inputs.

Strengthening of Opponent Processes by Repeated Stimulation

We have not yet explained the changes in hedonic dynamics brought about by repeated affect-arousing stimulations over a relatively long period of time. There are important differences between the A and B states on the left side of Table 1 as compared to the right side. Fortunately, one postulate brings order into the data: *The opponent process is strengthened through use and weakened through disuse,* but the primary affective process is *not* seriously affected by use. A *b* process will acquire more power if frequently elicited. It will show a shorter latency of response to *a,* a quicker rise, a higher asymptote, and a longer decay time. In contrast, an *a* process is a relatively stable, unconditioned reaction. This seems reasonable for a system which is designed to minimize deviations from affective neutrality. Why should an opponent process not act like a defensive or immunization process, which produces antibodies more efficiently and in larger numbers in the face of repeated challenge? In the same vein, *disuse* should weaken a *b* process, and it should slowly return to its original magnitude whenever its *a* process has not occurred for a long time.

Figure 4, Panel B, shows how the opponent processes will interact after the *b* process has been strengthened by repeated use. A comparison of Panels A and B explains why the left-hand portion of Table 1, describing "early stimulation," differs from the right-hand portion of Table 1, describing "later stimulations."

The effect of repeated experiences on the hedonic response is confined to strengthening the *b* process. During later stimulations the *b* process increases at a faster rate at stimulus onset and reaches a greater asymptotic intensity. In addition, the *b* process requires a much longer time to decay after the later stimulus terminations. As a consequence of these changes in the *b* process, the resultant pattern of the affective reaction changes so that the peak of A is considerably attenuated, and the peak of B becomes much greater and longer lasting. Figure 4, Panel B, shows these simple algebraic summations of the *a* and *b* processes after many repeated stimulations.

Figure 6 shows the pattern of emotional dynamics after many repeated stimulations and should be contrasted with the pattern shown in Figure 5. Three phenomena are corollaries of the *use postulate.* First, the peak of A′ will be less intense because the latency of the *b* process is decreased and its intensity is increased. Second, the steady level of A′, $|a - b|$, during maintained stimulation will be close to baseline and perhaps even below it in some cases. Third, the peak of B′ should be intense and long lasting, compared to what it was during early stimulation (Figure 5).

The events in Figure 6 do *not* follow from a simple *affective contrast* model. If a principle of simple hedonic or affective contrast were operating, then the peak of B in Figure 5 should be greater in intensity than it is in Figure 6, because the intensity of A directly prior to stimulus termination is greater in Figure 5 than it is in Figure 6. However, if we run down the descriptive adjectives for States A, B, A′, and B′ in Table 1, even as loosely defined as they are, the opponent-process model works well, and a simple contrast model fails.

More convincingly, the objective data from the electrocardiograph experiments with dogs fit the opponent-process model. In Table 1 we saw the following:

A, large acceleration;
B, small overshoot, short duration;

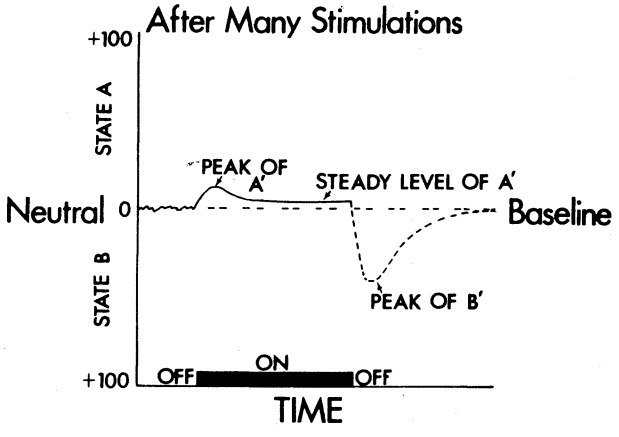

FIGURE 6. The manifest temporal dynamics generated by the opponent-process system after many repeated stimulations. (The major features of the modified pattern are labeled.)

A′, small or absent acceleration;
B′, large overshoot, longer lasting.

These findings fit the postulate that repetitions of affect-arousing stimulation, in this case shocks, will strengthen the opponent process which dampens the primary cardiac acceleration process. Furthermore, there are many signs of qualitative changes; e.g., signs of pleasure and euphoria appear in the period following termination of the shock session, although they were absent in earlier sessions. Our model cannot yet deal precisely with these qualitative changes which are produced by many repetitions of the same stimulus. A′ is less intense than A, and B′ is more intense and longer lasting than B. The model deduces these quantitative changes (Figure 4). In many cases, however, A′ is qualitatively different from A, even though they have the same hedonic sign. Similarly B′ and B are sometimes qualitatively different, even though they have the same hedonic sign. When the model is fully developed, it must contain a rationale for these qualitative changes.

The observations on the right-hand side of Table 1 also support the assumption that many repetitions of pleasures will strengthen their aversive opponent processes. Aversive states, manifesting themselves after the sudden termination of pleasurable inputs, become more intense with repeated experiences. Mild loneliness later becomes grief. Mild craving later becomes abstinence agony and intense craving. In addition, after many repetitions the steady level of pleasure produced by the continued presence of the pleasurable stimulus input has decreased. The confirmed opiate user experiences a "loss of euphoria," and the rush is gone. The pleasure-affective systems seem eventually to yield to opponent processes which keep departures from hedonic equilibrium relatively small. The aversive opponent process, when it is manifest, is more intense and longer lasting than it once was. So frequently repeated pleasure has its costs, psychologically, in an increased potentiality for displeasure.

Similarly, for A′ states aroused by aversive input, there is a "cost," but this cost is an increased potentiality for pleasure. Table 1 lists in the column under B′ the opponent affects for the aversive A′ states. Words like "joy," "exhilaration," and "good feeling" appear. These are the emotional costs of aversiveness. The model thus requires that any prolonged or repeated departures from hedonic or affective neutrality, regardless of hedonic sign, have a cost. Any significant departure from hedonic or affective neutrality should have correlates in increased autonomic and central nervous system activity aimed at reducing that departure. The cost of this activity will not only be psychological, but also will be physiological (metabolic, hormonal, and neural).

We are assuming that prolonged exercise of an opponent-process system, whether it be pleasurable or aversive, might cause physiological stress in the same sense that Selye (1950) uses the term stress. That is, many physiological resources might be required in order to keep the opponent process strong. If we follow Selye's argument, we would expect that such a constant demand might lead to the exhaustion of a particular overworked opponent-process system or to the debilitation of other defensive systems.

Thus we come to a major implication of our opponent-process model: There probably are stresses caused by pleasurable stimulation just as there are stresses caused by aversive stimulation. Therefore, there should be adaptational costs as a consequence of *both* kinds of stresses. Furthermore, if we look for them, we should find *diseases of adaptation for both,* due to the correlated, physiological side effects of long-duration often-elicited intense *b* processes.

In the past, psychologists have identified psychological stress with aversion, pain, and unpleasantness. Theories of mental disease, psychosomatic disease, and behavior disorders usually emphasize that aversiveness means stress, stress means aversiveness, and both cause emotional disorders, psychosomatic illnesses, and behavioral malfunctions. Our opponent-process theory of motivation puts all this to question. From our point of view, stresses caused by aversive stimulation are only one half of the story. There should be emotional disorders, psychosomatic diseases, and behavior disorders caused by long-lasting, repeated, and intense *b* processes in general, whether these opponent processes are pleasurable or aversive.

Take the case of the parachutists. Their operant behavior, when B′ is strong, will be reinforced by the pleasurable experience of B′. But the cost of neutralizing the innate aversiveness of a fall through space might be high, both physiologically and emotionally, just as Epstein (1967) has postulated. Even so, one might imagine, if there were no other pleasurable sensory inputs elsewhere available to the individual, that he might put himself through repeated aversive stimulation in order to experience the pleasurable B′ state which would be both intense and lasting. From our point of view, this type of apparent *masochistic* behavior is not a mental disorder but is, rather, a reflection of the *normal* functioning of a healthy, automatic, affect-control system. Thus, also, from this point of view, there is nothing abnormal or strange about addiction. It is only a socially vivid example of the normal hedonic and motivational functionings of an efficiently operating affect-control system.

Motivational systems involving pleasurable A states and aversive A states are similar. In both cases the onset, maintenance, and termination of the stimulus results in a certain amount of pleasure and a certain amount of displeasure. They differ mainly in whether

pleasure or displeasure comes first. In the case of the pleasurable A states, we can assume that the subsequent aversive B state functions as a drive that energizes the performance of operants, and that the pleasurable A states may positively reinforce these operants. Electrical self-stimulation of rewarding brain sites, chemical self-stimulation with opiates, and love relationships should work this way. In contrast, when the A state is aversive, and when the A state stimulation is absent, nothing functions as a drive to energize an operant upon which the A state is contingent. So we have the problem of how to get the behavior started. The B state for an aversive A state is not an energizer. Instead, it is a positive reinforcer. There is thus an important *asymmetry* between motivational systems for pleasurable and aversive A states. Some outside energizing influence is needed to get behavior started when the operant is followed by an aversive A state. Examples of such behaviors include "thrill-seeking" behaviors, such as parachute jumping, mountain climbing, automobile racing, etc., all of which involve an aversive component, followed by a pleasurable feeling of exhilaration. Why should one initiate an activity when its immediate effect is aversive, i.e., when a punishment contingency exists? Some competing outside influence, such as social pressure from peers, is required. However, after many repeated stimulations such outside influences may not be needed. Because the aversive A state is then weak and the subsequent, positively reinforcing B state is strong, the A state will function as a positive reinforcer. We know that this can happen when a weak shock signals the onset of food for cats (Masserman, 1943). Note that an outside influence is not needed when the A state is pleasurable. These operants are energized by the aversive B state.

Relation of the Theory to Other Concepts

To the reader well versed in the history of theories of learning, two aspects of our model should now be apparent. First, the phenomena of acquired motivation produced merely by the repetition of affect-arousing stimuli are nonassociative in nature. For example, the person repeatedly dosed with morphine does not have to know anything and is not required to be subjected to Pavlovian stimulus contingencies, nor to contingencies between operants and outcomes, in order to develop an increasing tendency to suffer when the morphine is withdrawn. The model is therefore very different from previous theories of acquired motivation, all of which have emphasized associational processes.

Second, the model, in relating its hypothetical mechanism to the phenomena of operant conditioning, explicitly assumes that operants are energized only by aversive states and that they can be reinforced either by the onset of pleasurable states or by the termination of aversive states. This is the case whether the pleasure or aversiveness comes from A states or B states. For example, the reinforcing effects of shock termination will not only be due to the elimination of the aversive A state but also, and perhaps more importantly, it will be due to the subsequent pleasurable B state. Woodworth and Schlosberg's (1954) concept of "safety," Mowrer's (1960) concept of "relief," and Denny's (1971) concept of "relaxation" are all emphasizing this concept of reinforcement in aversive situations.

The assumption that operants are energized only by aversive states fits most comfortably with the theoretical position taken by Hull (1943, 1952) and elaborated by Mowrer (1947), Miller (1948), Spence (1956), and Brown (1961). The assumption that operants can be reinforced by the onset of pleasurable states fits easily with the position taken by Young (1955) and by Pfaffman (1960). Of course, the assumption that operants are reinforced by the termination of aversive states is most like the drive-reduction position maintained especially by Hull (1943) and by Mowrer (1947). These assumptions are really not at issue for us. We take all three to be axiomatic, a point of departure, and we go on from there to spell out the dynamics of pleasurable and aversive states.

Conditionability of A States and B States

(not included in this reprinting)

Analysis of Selected Motivational Phenomena in Light of the Opponent-Process Theory

(not included in this reprinting)

A States Which Have Little or No Opponent Process

A few hedonic disequilibriums may cause very little corrective, oppositional reaction in the nervous system. A nonopposed system would manifest no peak of the A state, no adaptation, and no appearance of a B state after stimulus termination. One possible example is the hedonic state engendered by marijuana. As a chemical stimulus it precipitates a mildly pleasurable A state. However, there is no reported peak or adaptation, nor are there aversive withdrawal symptoms or craving. Furthermore, tolerance often does not develop with repeated dosages. The concommitance of *all* these attributes would be exactly what the opponent-process model would predict for a pleasure without an opponent. Nausea is an example of an aversive A state which may have no opponent B state. Perhaps some aesthetic pleasures have no opponent process.

We have already talked about the variations in the intensity and duration of B states as a function of the particular kind of A state elicited. For example, we

mentioned the intense and long-lasting aversive B states in the cases of grief and opiate withdrawal. In contrast, we mentioned the relatively short duration of B states associated with the taste of M & Ms and peanuts, and rewarding electrical stimulation of the brain. In addition to these inherent variations in intensity and duration of B states elicited by different types of stimuli, repeated elicitation causes large changes in the intensity and duration of B states in some cases and not in others. Therefore, differences in the initial strength of *b* processes must reflect a parameter of our model, and the effects of repeated elicitation of A states must operate on this parameter. In the case of A states having no opponent process, the value of the parameter is zero.

We have been constantly amazed by the huge variations in the strengths and durations of different *b* processes after repeated elicitations. Some *b* processes, even when well exercised, last but a few minutes. Some examples are taste cravings, the average *b* process for electrical stimulation of the brain, cardiac deceleration after shock termination, the "fit of joy" when the dog is released from a shock box, etc. Some last a few hours. Examples are the exhilaration following dangerous or endurance-challenging exercise, or distress calling in precocial birds when the imprinting object is gone. Yet others last for months. Examples are loneliness and grief, craving following withdrawal from either opiates, alcohol, or barbiturates.

At first glance it may appear that we have completely undercut our theory. We have speculated that some A states may not arouse B states, and we have provided no principles by which one could designate in advance whether or not a particular A state would be a part of an opponent-process pair. At present, we look on this as an empirical problem, and we use the following argument. Opponent processes defend a hedonic equilibrium. They are part of the biological defense system mediated by the brain. In the realm of foreign body reactions to bacteria, viruses, and poisons we often can detect defense systems. Indeed, the actions of antigens and antibody formation bear many resemblances to the opponent-process systems we have described. However, not all poisons and not all foreign bodies engender defense reactions. We can be defenseless. These substances are, therefore, called deadly poisons. We think the same situation holds in the defense of hedonic equilibrium, and so we should not be surprised to discover that a given A state goes relatively unopposed. Perhaps one day we will have a theoretical rationale for the parameter and operator involved here.

Discussion

We have argued that there are certain systems in the brain, the business of which is to suppress or reduce all excursions from hedonic neutrality, whether those excursions be appetitive or aversive, pleasant or unpleasant. The systems operate to decrease the intensity of subjective "hedonic quality," "affect," "emotion," "arousal," or the objective reinforcing properties of stimuli. The systems function independently of operants or instrumental acts. They are fully automatic. Thus, whereas operants tend to maximize positive reinforcement and to minimize negative reinforcement, the affect-controlling systems of the central nervous system minimize both. They are brought into play whenever significant departures from affective equilibrium occur as a consequence of stimulation onset and maintenance. When such a system is effective, it will reduce the intensity of the affective experience even while the input is still there. This reduction will be manifest for both positive and negative reinforcers, and for pleasant and unpleasant stimuli. The theoretical model refers to both subjective and objective psychological phenomena.

Reduction in affective or hedonic intensity is postulated to be brought about by the activation of an opponent loop, precipitated into action whenever affective, hedonic, or emotional states are aroused. The opponent loop opposes the stimulus-aroused affective state. Furthermore, the opponent process is postulated to be sluggish in its latency, recruitment, and decay (a heavily damped circuit, an inertia-laden system). The opponent loop itself is postulated to generate an hedonic process which is, in some abstract sense, the opposite to that precipitated by the stimulus input which has initially aroused affect. The opponent process will manifest its quality and intensity when the stimulus is suddenly terminated. The persistence of the opponent process will be seen for some time because of its sluggish decay property.

Furthermore, we have postulated that the opponent process is strengthened through use and weakened through disuse. These changes are nonassociative in nature. This makes the affect-control systems similar to some immunological mechanisms in their properties.

Even though the model is not yet as precise as some mathematical models, from it, nevertheless, one can unambiguously deduce many of the known phenomena of acquired motivation. As examples of this, we have illustrated in detail how the model organizes information on aversive behavior control and the drug addictions.

The theory postulates no conditioning or learning mechanisms responsible for the occurrence of the acquired motivations we have discussed. The acquisition is automatic, merely by virtue of the repeated occurrence of affect-producing stimuli. However, the interrelationships between the affect-control mechanisms and Pavlovian conditioning processes, and the interrelations of both with operant behavior, are discussed in the sections not included in this reprinting.

The novel feature of the opponent-process theory is that it sees the behavioral phenomenon of addiction as

an empirical model for all acquired motivation. Addiction is not viewed as an abnormality. Instead, it is the inevitable consequence of a normally functioning system which opposes affective or hedonic states. We assume, for example, that love is an addiction phenomenon characterized by habituation to the presence of the loved one and intensified aversion in the absence of the loved one. In the same vein, we assume that imprinting in precocial birds is an addiction phenomenon. In the case of aversive stimulation, we assume that masochistic phenomena are the consequence of a normally functioning system which opposes affect. These phenomena are characterized by habituation to the presence of the feared or unpleasant event and intensified pleasure after the termination of that event.

Finally, we have pointed out some new lines of empirical research suggested by the opponent-process theory of motivation.

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